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Valve prosthesis—patient mismatch
A long-term sequela

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SUMMARY A patient underwent mitral valve replacement in 1966 for severe mitral valve disease. The initial clinical result was excellent but symptoms recurred nine years later, and since 1969 he has had progressive cardiac enlargement. At present he is in functional class II and has massive cardiomegaly with gross enlargement of the left atrium, right ventricle, and right atrium. Cardiac catheterisation showed "normal" prosthetic valve function, but on moderate exercise he developed severe left atrial and pulmonary arterial hypertension. His clinical course illustrates a long-term sequela of "valve prosthesis–patient mismatch".

The problem of prosthetic valve—patient mismatch has recently been emphasised[1]; the long-term consequences of this mismatch, however, are not known. We describe a patient with "normal" prosthetic mitral valve function who, over the years, developed gross cardiomegaly.

Case report

A 20-year-old man developed rheumatic fever in 1964. He had no history of previous rheumatic episodes. Within a few months he developed weakness, light-headedness, and congestive heart failure. He was digitalised and a diagnosis of rheumatic heart disease was made. In April 1966 there was progressive dyspnoea on exertion, intermittent paroxysmal nocturnal dyspnoea, increasing weight gain, and haemoptysis. Clinical examination showed that he was in "heart failure", with signs of severe mixed mitral valve disease. The patient was

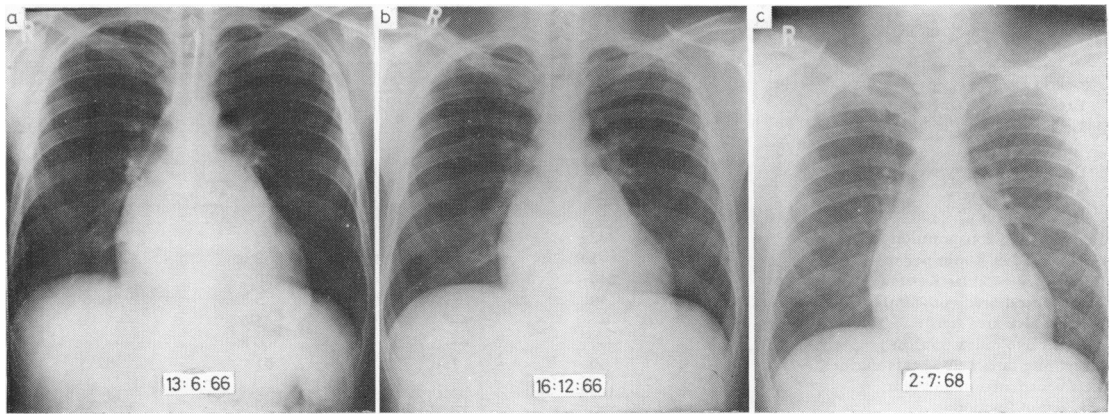

Fig. 1 *Preoperative chest x-ray film, June 1966 (a), showing cardiomegaly with enlargement of the left ventricle and left atrium; six months after operation there is some reduction in heart size; and two years later (c) there is enlargement of the left atrium.*

in New York Heart Association functional class III. Electrocardiogram showed atrial fibrillation with a controlled ventricular rate and left ventricular hypertrophy. Chest *x*-ray showed cardiomegaly with left atrial enlargement (Fig. 1). Cardiac catheterisation (Table) in June 1966 confirmed the diagnosis of severe mixed mitral valve disease. In August 1966, he successfully underwent mitral valve replacement with Starr-Edwards prosthesis 4M–6120 (32 mm, Silastic ball, non-cloth covered mitral prothesis). The tricuspid valve was normal. Clinically the result was good and he returned to functional class I. He was given digoxin to control his ventricular rate and continuous warfarin because of his prosthetic valve (Fig. 1). He continued to work as a plywood mill worker and remained active.

In 1975, he noted the onset of some mild dyspnoea on exertion that progressed slowly though he remained active and was working full time. He was referred back to our medical centre in 1978 because of increasing dyspnoea. He could walk on the level at a regular pace as long as he liked and was able to climb five flights of stairs before becoming dyspnoeic. He did not complain of orthopnoea, paroxysmal nocturnal dyspnoea, chest pain, ankle swelling, or dizziness, and did not have a history of embolic episodes. Physical examination showed that all peripheral arterial pulses were present and equal. Blood pressure was 120/80 mmHg. His height and weight were 165 cm (5′ 9″) and 78 kg (172 lb), respectively (BSA 1·94 m^2). The lungs were clear to auscultation and percussion. The estimated jugular venous pressure was 14 cm of water. The cardiac impulse was displaced 2 cm to the left of the midclavicular line in the sixth intercostal space. There was a right ventricular heave. The prosthetic opening and closing sounds were normal and he had a grade 1/6 apical ejection systolic murmur. There was no hepatosplenomegaly and no peripheral oedema. The electrocardiogram showed atrial fibrillation with a controlled ventricular rate. The mean QRS axis in the frontal plane was +120°, and there was non-specific T wave flattening in the praecordial leads. Chest *x*-rays (Fig. 2) showed gross cardiomegaly with enlargement of the left atrium, right ventricle, and right atrium. Review of the chest *x*-rays since 1966 (Fig. 1 and 2) showed that he had progressive enlargement of these three chambers, now massively dilated. M-mode echocardiogram showed left atrial systolic dimension of 9·5 cm and left ventricular diastolic dimension of 6·5 cm. There was no pericardial effusion. A treadmill exercise test was performed using the Bruce protocol. He was limited at 15 seconds into stage IV because of leg fatigue. There were no ST or T wave changes at peak exercise heart rate of 180 beats/min. Cardiac catheterisation showed a raised right atrial mean pressure at rest and there was no gradient across the tricuspid valve. Left atrial and pulmonary artery pressures were in the upper range of normal at rest.[2] On exercise, however, there was a pronounced rise in left atrial and pulmonary arterial pressures (Table). The resting mean gradient of 5·7 mmHg at a heart rate of 51 beats per minute and a cardiac index of 2·6 l/min per m^2 was in the "normal" range,[3 4] as was the calculated prosthetic

Table

	Before valve replacement		*After valve replacement*	
	Rest	*Exercise*	*Rest*	*Exercise*
Pressures (mmHg)	*16 June 1966*		*4 April 1978*	
Brachial artery (mean)	140/80 (85)	170/95 (105)	138/84 (100)	170/84 (122)
Left ventricle	140/12	175/9	144/12	170/18
Left atrium	—	—	V20 (14)	V57 (34)
Pulmonary artery wedge	V16 (13)	V60 (41)	—	—
Pulmonary artery	36/17 (23)	100/54 (70)	37/15 (18)	84/33 (48)
Right atrium	(5)	—	V=14 (9)	—
Body oxygen consumption (ml)	232	1188	298	1046
Cardiac index (l/min per m^2)	1·9	4·9	2·6	5·2
Pulmonary vascular resistance (dynes s cm^{-5})	240	256	120	104
Mean mitral gradient (mmHg)	8	20	5·7	14·5
Mitral valve area (cm^2)	—	—	1·6	—
Mitral valve index (cm^2/m^2)	—	—	0·8	—
Heart rate (atrial fib; beats/min)	60	156	51	87
Left ventricular angiogram				
Ejection fraction	—	—	0·65	—
End-diastolic volume index (ml/m^2)	—	—	134	—
	Moderate mitral regurgitation No aortic regurgitation		No mitral regurgitation No aortic regurgitation	

area of 1·6 cm. The left ventricle was mildly enlarged, and the ejection fraction was normal. On angiography, there was no evidence of mitral regurgitation or of aortic regurgitation. In the past year, he has remained symptomatically unchanged and is being treated with digoxin, warfarin, and oral penicillin as prophylaxis against recurrent rheumatic fever.

Discussion

What is the cause of recurrence of symptoms and of increasing heart size after mitral valve replacement? He has taken warfarin regularly, has had no thromboembolic episodes, and the prosthetic valve area was in the "normal" range. Since the pulmonary vascular resistance was normal at rest and on exercise both before and after operation, incomplete regression or progression of pulmonary vascular disease would not explain the observed changes in the right heart. There was no mitral regurgitation and no evidence for aortic valve disease; and though the left ventricular end-diastolic volume index was mildly increased, left ventricular systolic pump function was normal. The increased left ventricular end-diastolic volume index probably represents a residuum of his previous mitral regurgitation. Though coronary arteriography was not performed, this 35-year-old man has never had angina and maximal exercise stress testing did not show evidence of myocardial ischaemia. The existence of subclinical coronary artery disease is not disproven; even if this were present, however, it would not explain the observed abnormal haemodynamics on exercise.

Recently, we have emphasised the problem of "valve prosthesis patient mismatch",[1] resulting from two factors. First, the *in vivo* effective prosthetic valve area of almost all types of valve replacement devices that can be inserted in most patients is less than that of the normal human valve. The *in vivo* effective prosthetic valve area is even further reduced because of tissue ingrowth and endothelialisation, and, therefore, these devices can be considered "stenotic". Second, in some patients the problem is compounded because the size of the

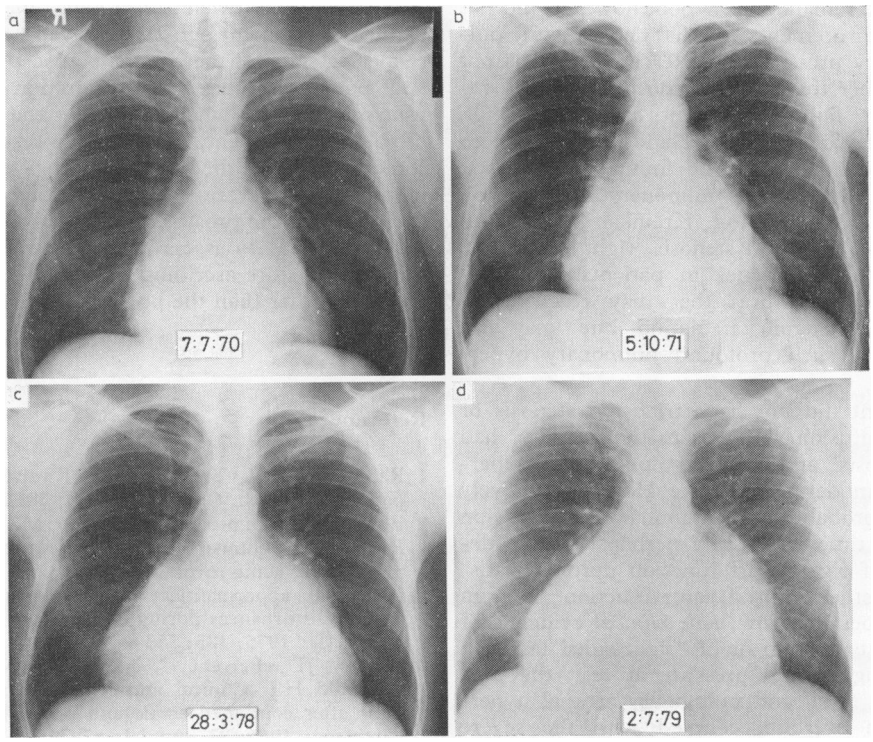

Fig. 2 *Chest x-ray film in 1970 showing (a) cardiomegaly, considerable enlargement of the left atrium, and probable enlargement of the right atrium; in 1971 (b), 1978 (c), and 1979 (d) progressive cardiomegaly with massive enlargement of the left atrium and considerable enlargement of the right heart is present.*

prosthesis that can be inserted is limited by the size of the annulus, which is small compared with the size of the patient, and also by the size of the cavity in which the prosthesis must lie. Usually, the obstruction is mild to moderate, but at times, it is moderate to severe. The patient we are reporting has "normal" prosthetic valve function, with the haemodynamics of moderate (to severe) mitral stenosis. The natural history studies of mitral stenosis[5 6] emphasise that there is a "latent" period of mitral stenosis often made up of the stage of formation of mitral stenosis and, after that, an asymptomatic stage, even though fully developed mitral stenosis is present. In about half of the patients, symptoms develop gradually, and in the other half are often abruptly precipitated by such complications as atrial fibrillation. In his classic work on mitral stenosis, Paul Wood[5] observed that "when the shadow of the right side of the heart was conspicuously enlarged and the pulmonary artery normal or only slightly dilated, tricuspid stenosis was present in 44%, pericardial effusion in 12·5%, and "congestive heart failure" without an unduly high pulmonary vascular resistance in 25%; no explanation other than auricular fibrillation could be found for the remainder". Wood, however, did not describe measurements of left atrial and pulmonary artery pressures on exercise, and it is not known whether the patients who developed "congestive heart failure" without an unduly high pulmonary vascular resistance and those in whom an explanation could not be found had, in fact, moderate or even severe pulmonary hypertension on exercise. Bristow and Kremkau[7] have also observed that, in mitral stenosis, right ventricular failure usually supervenes in patients with pulmonary artery pressures that may reach levels found in the systemic circulation, but "may also occur without such profound pulmonary hypertension".

Our patient did not have tricuspid stenosis or pericardial effusion, but, haemodynamically, had "mitral stenosis" and had severe pulmonary hypertension on moderate exercise. He was relatively active, and probably had pulmonary hypertension most of the time, producing perhaps his late deterioration of right heart function despite "satisfactory" prosthetic mitral valve function. Though at rest he does not now have *clinical* evidence of tricuspid regurgitation or of "right-sided failure", the raised right atrial pressure at rest, the pronounced left atrial and pulmonary arterial hypertension with exercise, coupled with the severe progressive enlargement of his left atrium, right ventricle, and right atrium suggest that his clinical course will continue to be similar to that of some patients with moderate to severe mitral stenosis whose eventual clinical symptoms are predominantly those of "right-sided heart failure". Clearly a much larger effective prosthetic valve area would be desirable to help this patient. Since all prosthetic valves have a reduced effective orifice area and he has a prosthetic valve area that is "satisfactory" and, moreover, since there is no guarantee that another prosthesis will give him a much larger prosthetic valve area or that he will be free of other complications of prosthetic valves, his physician has decided to treat himmedically.

CLINICAL IMPLICATIONS

This patient shows that "valve prosthesis—patient mismatch" can cause late deterioration of cardiac function. His case emphasises that: (1) an effective prosthetic valve area that is acceptable for a small inactive patient can be unsatisfactory for a larger physically active individual. Therefore, the calculated absolute effective prosthetic orifice size needs to be corrected for body size and the function of the prosthetic valve needs to be studied, not only at rest, but also at a different haemodynamic state—particularly exercise; (2) though patients may remain clinically "well" for a long time, haemodynamic derangement persists after valve replacement rendering some of these patients susceptible to late, and perhaps "sudden", deterioration of cardiac function; and (3) when evaluating the late results of valve replacement, the poor late results may, in some patients, be the outcome of delayed effects of moderate (to severe) prosthetic valve "stenosis" (even though the prosthesis is "normal") therefore, it is important to assess prosthetic valve function accurately before ascribing undesirable late results to causes other than the prosthetic valve.

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